



Fig. 1. The anterior view of the leftward rudimentary right ventricle from a heart with double-inlet left ventricle, showing the segment of apical trabecular septum that can safely be removed to enlarge the ventricular septal defect (VSD) without damaging the atrioventricular bundle.

are also discussed at length in the book *Double Inlet Ventricle*.³

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12/8/72318

Reply to the Editor:

We have read the letter from Dr. Anderson regarding the location of the atrioventricular conduction bundle in {S,L,L} hearts and are in complete agreement with the location of the bundle in these hearts. In fact, we acknowledged Dr. Anderson's excellent work in this area in our article.

We wish to emphasize, however, that we are not confused about the location of the conduction system. The statement that we made in the article that "the non-branching bundle courses at the superior margin of the BVF [bulboventricular foramen]" refers to the anatomy when viewed from the transatrial approach, that is, from the left ventricular side. This is our preferred approach for examining the BVF, because it is the least destructive.

We are in complete agreement with Dr. Anderson that sometimes it is possible to resect a wedge of tissue from the apical trabecular septum by incising close to the obtuse margin of the ventricular mass. However, more extensive resection is often needed in severely restrictive BVFs, and this is especially more destructive in neonates and small infants. In such patients we prefer to perform a Damus procedure. Furthermore, the intraoperative manipulation necessary to expose the BVF through the rudimentary chamber, the great vessels, or the atrioventricular valve results in substantial distortion. When this problem is combined with the intrinsic fragility of the bundle in patients with {S,L,L} hearts, the risks of conduction injury are high.

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12/8/72319

Surgical technique and atrial arrhythmias after total cavopulmonary connection

To the Editor:

We read with much interest the report by Hashimoto and associates¹ in the September 1995 issue of the Journal, in which a method of total cavopulmonary connection with an autogenous intraatrial tunnel was presented. Postoperative atrial arrhythmias are a widely recognized and very troublesome clinical entity after various forms of Fontan repair.² We have several questions pertaining to the occurrence of postoperative atrial arrhythmias after the technique described in this article.

The authors state that "preserving the crista terminalis and the sinus node and its arteries" prevented the development of postoperative atrial arrhythmias. Sinus node dysfunction and conduction abnormalities related to the crista terminalis have been linked to the genesis of atrial arrhythmias, both in patients who have had the Fontan operation^{3,4} and in naturally occurring human atrial flutter.⁵ Because we are presently working with an experimental model to elucidate more clearly the electrophysiologic importance of these factors with respect to atrial flutter after the Fontan operation, we are very interested to know whether the authors based their conclusions on